Lean body mass is the independent and predominant anthropometric variable associated with atrioventricular block

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BACKGROUND Information regarding the association between lean body mass (LBM) and atrioventricular (AV) block risk is lacking.

OBJECTIVE The purpose of this study was to determine the associations of LBM with the risk of AV block.

METHODS We analyzed 370,415 UK Biobank participants who underwent bioimpedance analysis and had no history of AV block or cardiac implantable electronic devices. The primary outcome was a composite of second- or third-degree AV block. Secondary outcomes included each component of the primary outcome and AV block–related pacemaker implantation. Adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) per 1-SD increase in LBM were estimated using multivariable Cox regression.

RESULTS The mean age was 56.8 years, and 52.4% were women. During a median follow-up of 11.8 years, 1519 (0.9%) and 677 (0.4%) primary outcome events occurred in men and women, respectively. LBM per 1-SD increase (7.7 kg in men and 5.0 kg in women) was associated with a 15% (HR 1.15; 95% CI 1.09–1.21; $P < .001$) and 13% (HR 1.13; 95% CI 1.05–1.22; $P = .001$) higher risk of the primary outcome in men and women, respectively. Associations remained significant even after adjustment for various anthropometric variables. Similar patterns were observed for all secondary outcomes. Higher LBM was associated with a prolonged PQ interval. The sex difference in incident primary outcome was attenuated after LBM adjustment.

CONCLUSION Higher LBM was associated with an increased AV block risk in both sexes and with a higher prevalence of a prolonged PQ interval. Sex differences in AV block risk may be partly mediated by differences in LBM.

KEYWORDS Atrioventricular block; Electrocardiography; Lean body mass; Pacemaker implantation; PQ interval

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Introduction

As older age is related to a higher risk of atrioventricular (AV) block, the incidence and prevalence of AV block are expected to increase in an aging society.[1,2] Pacemaker implantation is an effective treatment for AV block; however, it may lead to device-related complications.[3] Cardiac conduction disorders are not typically considered preventable; however, recent studies have identified potentially modifiable risk factors, including systolic blood pressure, fasting glucose level, metabolic status, and physical activity.[1,4,5] Thus, identifying risk factors for AV block, particularly modifiable risk factors, is important.

Previous studies have demonstrated that obesity, commonly defined by body mass index, is associated with a higher risk of incident AV block.[6–8] Body mass index is therefore often regarded as a modifiable risk factor; however, it is a crude anthropometric measure that does not distinguish between lean body mass (LBM) and fat mass and may not accurately reflect an individual's adiposity.[9] Thus, body mass index–based associations do not clarify which component of body composition underlies the observed risk. Accordingly, evaluating whether LBM, which consists primarily of skeletal muscle, or fat mass is associated with an increased risk of AV block is necessary to identify truly modifiable components relevant to incident AV block. To our knowledge, no previous study has reported the association between LBM and the risk of AV block. Given this gap, we hypothesized that LBM may serve as an independent and predominant anthropometric risk factor for AV block. The first objective of this study was to examine the association between LBM and the risk of AV

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KEY FINDINGS

- In this UK Biobank prospective cohort study, increased lean body mass (LBM) was associated with a higher risk of second-degree atrioventricular (AV) block, third-degree AV block, and AV block–related pacemaker implantation in both men and women. Associations remained robust even after adjustment for various anthropometric variables, including fat mass.
- Increased LBM was also associated with a higher prevalence of a prolonged PQ interval (>200 ms), suggesting a relationship between LBM and AV conduction delay.
- The sex difference in the risk of the composite of second- or third-degree AV block was attenuated after adjusting for LBM, suggesting that sex differences in AV block risk may be partly mediated by differences in LBM.

block, defined as a composite of second- or third-degree AV block. The second objective was to assess the relationship between LBM and electrocardiographic parameters to better understand the potential mechanisms linking LBM and AV block risk.

Methods

Ethical approval

The UK Biobank study received ethical approval from the North West–Haydock Research Ethics Committee (REC reference: 21/NM/0157). All participants provided informed consent, and those who withdrew consent were excluded from the analysis. The present study was conducted under UK Biobank application no. 77793 and approved by the Institutional Review Board of the Yonsei University Health System (approval no. 4–2025–0652), with a waiver of additional informed consent. The research reported in this article adhered to the Declaration of Helsinki.

Study population

We conducted a prospective cohort study using data from the UK Biobank, which recruited 502,421 middle-aged participants (aged 40–69 years) across 22 assessment centers in England, Scotland, and Wales between 2006 and 2010.[10] At baseline, detailed health-related data were collected through interviews, questionnaires, physical measurements including whole-body bioimpedance analysis, and blood sampling. We included 370,415 participants who underwent whole-body bioimpedance analysis and had no history of second- or third-degree AV block or cardiac implantable electronic devices. The sample selection flowchart is presented in Supplemental Figure 1.

Assessment of LBM

LBM was assessed using whole-body bioimpedance analysis (Tanita BC-418MA body composition analyzer [Tanita Corp., Tokyo, Japan]) under standardized protocols at baseline assessment centers. Specifically, LBM was defined on the basis of the whole-body fat-free mass (UK Biobank data field: 23101), corresponding to weight minus whole-body fat mass. Complementary measures of weight, whole-body fat mass, and body fat percentage were obtained from the UK Biobank data fields 23098, 23100, and 23099, respectively.

Definition of outcomes and covariates

Participants' history of second- or third-degree AV block and cardiac implantable electronic device implantation were identified using linked UK Biobank hospital inpatient data with *International Classification of Diseases, Tenth Revision* (*ICD-10*) and *OPCS Classification of Intervention and Procedures, version 4* (*OPCS-4*) codes. Incident AV block events were ascertained from the UK Biobank hospital inpatient and national death registry data, with censoring at the first event, loss to follow-up, or death.

The primary outcome was a composite of second-degree or unspecified AV block (*ICD-10* codes I44.1 or I44.3) or third-degree (complete) AV block (*ICD-10* codes I44.2, I45.8 [AV dissociation], or I45.9 [heart block not otherwise specified or Stokes-Adams syndrome]). Secondary outcomes included each component of the primary outcome and AV block–related pacemaker implantation (*OPCS-4* procedure codes K605, K606, K615, and K616, identified following *ICD-10* codes I44.1, I44.2, I44.3, I45.8, and I45.9). Supplemental Methods provide detailed information on the covariates used in the analysis, and Supplemental Table 1 lists the definitions of outcomes and comorbidities.

Primary analysis

We performed a complete-case analysis, excluding participants with missing covariate data (n = 120,498 [24.5%]). After confirming the markedly different distributions of LBM between men and women, participants were stratified by sex, and all analyses were performed separately to account for sex-specific differences. Participants were followed from the baseline assessment until the first occurrence of the primary or secondary outcomes, loss to follow-up, or death, whichever occurred first. Associations between LBM (per 1-standard deviation [SD] increase) and the risk of outcomes were evaluated using multivariable Cox proportional hazards models. Covariates for adjustment were selected a priori or on the basis of a univariable association with outcomes ($P < .10$). Adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) were estimated after adjustment for age, white ethnicity, hypertension, diabetes mellitus, dyslipidemia, coronary heart disease, heart failure, atrial fibrillation, chronic kidney disease, sleep apnea, hypothyroidism, hyperthyroidism, smoking and alcohol history, high-sensitivity C-reactive protein, physical activity,

Townsend deprivation index, education attainment, and antiarrhythmic drug use. The proportional hazards assumption was assessed using Schoenfeld residuals and the Grambsch-Therneau test. *E* values were calculated to estimate the minimum strength of association that an unmeasured confounder would require to explain away the observed association.

Additional anthropometric analyses

To determine whether LBM represented the independent and predominant anthropometric variable associated with the primary outcome, we analyzed LBM and 8 anthropometric traits (fat mass, fat percentage, height, weight, body mass index, waist circumference, hip circumference, and waist-to-hip ratio), both individually and in pairwise models with mutual adjustment, with adjustment for covariates included in the main analysis.

Restricted cubic splines were fitted to visualize the dose-response relationships of LBM or fat mass (per 1-SD increase) with the HR for the primary outcome, adjusted for covariates included in the main analysis and mutually adjusted for fat mass or LBM. 3 knots were selected on the basis of the lowest Akaike information criterion value. The reference value was defined as the mean LBM for each sex.

Exploratory analyses

To explore potential mechanisms underlying the association between LBM and the risk of the primary outcome, we examined trends in the prevalence of a prolonged PQ interval (>200 ms) and prolonged QRS duration (>120 ms) across quartiles of LBM using linear regression.

To evaluate whether the difference in LBM between men and women contributes to the sex-related difference in the risk of the primary outcome, we divided the crude incidence rate of the primary outcome in men by the estimated HR corresponding to the sex difference in LBM and visually compared it with that in women.

Sensitivity analyses

Several sensitivity analyses were performed. First, we assessed the correlation between bioimpedance-derived and dual-energy X-ray absorptiometry (DXA)–derived LBM among 34,638 participants (16,827 men and 17,811 women) with available DXA measurements to validate bioimpedance-derived LBM and address potential misclassification bias. Second, we repeated the main analysis using multiple imputation by chained equations (predictive mean matching for continuous variables and logistic regression or multinomial logistic regression for categorical variables) to address missing data. Third, participants with atrial fibrillation or antiarrhythmic drug use at baseline were excluded, and the main analyses were repeated, as AV conduction may not be reliably assessed in their presence. Fourth, as both systolic and diastolic blood pressures are associated with AV block, we performed the main analyses with adjustment for both systolic and diastolic blood pressures instead of hypertension.[7] Fifth, to minimize reverse causality, participants who developed the primary outcome within 2 years of baseline were excluded, and the main analysis was repeated. Sixth, to account for potential changes in cardiovascular disease status during follow-up, we additionally adjusted for myocardial infarction or heart failure as time-updated covariates and repeated the main analysis. Seventh, we additionally adjusted for resting heart rate to evaluate whether vagal tone drives the association between LBM and the primary outcome.

Statistical analysis

Continuous variables are presented as mean ± SD or median (interquartile range) and categorical variables as count (percentage). The Student *t* test or the Mann-Whitney *U* test was used to compare continuous variables, and the χ^2 test or Fisher exact test was used for categorical variables. 2-tailed *P* values <.05 were considered statistically significant. All statistical analyses were performed using R software version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Baseline characteristics

Among the 370,415 UK Biobank participants included in the study, the mean age was 56.8 ± 8.1 years and 52.4% were women. The mean LBM was significantly higher in men than in women (63.7 ± 7.7 kg vs 44.5 ± 5.0 kg; $P <$ .001) (Supplemental Figure 2). Baseline characteristics of the study sample, stratified by sex, are presented in Table 1. The baseline characteristics of UK Biobank participants included in and excluded from the study sample are compared in Supplemental Table 2. Included participants were more likely to be younger and male. However, there were no notable differences in race or comorbidities (standardized mean difference <0.100 for all comparisons).

LBM and primary and secondary outcomes

During a median follow-up of 11.8 years, 1519 (0.9%) and 677 (0.4%) primary outcome events occurred in men and women, respectively. The incidence of the primary outcome was 0.75 cases per 1000 person-years in men and 0.30 cases per 1000 person-years in women. LBM per 1-SD increase was associated with a 15% (HR 1.15; 95% CI 1.09–1.21; $P <$ .001) and 13% (HR 1.13; 95% CI 1.05–1.22; $P =$ .001) higher risk of the primary outcome in men and women, respectively (Table 2). Older age, higher high-sensitivity C-reactive protein levels, and a history of cardiovascular risk factors and cardiovascular disease were also associated with an increased risk of the primary outcome in both sexes (Supplemental Tables 3 and 4).

Associations between LBM and secondary outcomes followed a similar trend to that of the primary outcome. LBM per 1-SD increase was associated with a higher risk of second-degree AV block (HR 1.16; 95% CI 1.08–1.26; $P <$ .001 in men; HR 1.13; 95% CI 1.01–1.27; $P =$ .031 in

Table 1 Baseline characteristics of the study sample stratified by sex

Variable	Overall (N = 370,415)	Men (n = 176,350)	Women (n = 194,065)
Age (y)	56.8 ± 8.1	57.1 ± 8.2	56.5 ± 8.0
Race			
White	350,948 (94.7)	167,082 (94.7)	183,866 (94.7)
Others*	19,467 (5.3)	9268 (5.3)	10,199 (5.3)
Height (cm)	168.9 ± 9.3	175.8 ± 6.8	162.7 ± 6.3
Weight (kg)	78.2 ± 15.8	85.9 ± 14.0	71.1 ± 13.8
Lean body mass (kg)	53.7 ± 11.6	63.7 ± 7.7	44.5 ± 5.0
Fat mass (kg)	24.5 ± 9.4	22.2 ± 8.1	26.6 ± 9.9
Fat percentage (%)	31.0 ± 8.4	25.2 ± 5.7	36.3 ± 6.9
Body mass index (kg/m^2)	27.3 ± 4.7	27.8 ± 4.1	26.9 ± 5.0
Waist circumference (cm)	90.1 ± 13.3	96.7 ± 11.1	84.2 ± 12.3
Hip circumference (cm)	103.2 ± 8.9	103.4 ± 7.4	103.0 ± 10.1
Waist-to-hip ratio	0.9 ± 0.1	0.9 ± 0.1	0.8 ± 0.1
Smoking history			
Never or previous	332,779 (89.8)	155,433 (88.1)	177,346 (91.4)
Current smokers	37,636 (10.2)	20,917 (11.9)	16,719 (8.6)
Alcohol history			
Never or previous	27,469 (7.4)	10,390 (5.9)	17,079 (8.8)
Current drinkers	342,946 (92.6)	165,960 (94.1)	176,986 (91.2)
Physical activity (MET min/wk)†	1618.7 ± 2060.9	1765.9 ± 2272.3	1484.9 ± 1837.9
Townsend deprivation index‡	−1.4 ± 3.0	−1.4 ± 3.1	−1.4 ± 3.0
Education attainment§			
ISCED category 1	56,719 (15.3)	27,771 (15.7)	28,948 (14.9)
ISCED category 2	97,628 (26.3)	41,968 (23.8)	55,660 (28.7)
ISCED category 3	43,261 (11.7)	18,955 (10.8)	24,306 (12.5)
ISCED category 4	18,817 (5.1)	7828 (4.4)	10,989 (5.7)
ISCED category 5	153,990 (41.6)	79,828 (45.3)	74,162 (38.2)
Comorbidities			
Hypertension	103,198 (27.9)	57,639 (32.7)	45,559 (23.5)
Diabetes mellitus	18,225 (4.9)	11,841 (6.7)	6384 (3.3)
Dyslipidemia	51,963 (14.0)	31,473 (17.8)	20,490 (10.6)
Coronary heart disease	10,224 (2.8)	8057 (4.6)	2187 (1.1)
Heart failure	8287 (2.2)	4081 (2.3)	4206 (2.2)
Atrial fibrillation	4712 (1.3)	3487 (2.0)	1225 (0.6)
Chronic kidney disease	3529 (1.0)	1869 (1.1)	1660 (0.9)
Sleep apnea	2122 (0.6)	1685 (1.0)	437 (0.2)
Hypothyroidism	10,302 (2.8)	1884 (1.1)	8418 (4.3)
Hyperthyroidism	1563 (0.4)	364 (0.2)	1199 (0.6)
Medications			
Antiarrhythmic drugs	887 (0.2)	574 (0.3)	313 (0.2)
High-sensitivity CRP (mg/L)	1.3 (0.6–2.6)	1.2 (0.6–2.5)	1.3 (0.6–2.8)

Values are presented as mean ± standard deviation, median (interquartile range), or n (%).

CRP = C-reactive protein; ISCED = International Standard Classification of Education; MET= metabolic equivalent of task.

*Other races consist of Asian, black, mixed, and others/unknown.

†Only MET min/wk corresponding to moderate-to-vigorous physical activity was included in the analysis of physical activity.

‡Positive values of the Townsend deprivation index indicate high material deprivation, whereas negative values indicate relative affluence.

§A higher ISCED category indicates more years of education.

women), third-degree AV block (HR 1.14; 95% CI 1.06–1.22; $P < .001$ in men; HR 1.15; 95% CI 1.04–1.26; $P = .005$ in women), and AV block–related pacemaker implantation (HR 1.17; 95% CI 1.09–1.25; $P < .001$ in men; HR 1.19; 95% CI 1.08–1.31; $P < .001$ in women) (Table 2).

Anthropometric variables and the primary outcome

All anthropometric variables were associated with a higher risk of the primary outcome in men, whereas only weight, waist circumference, and waist-to-hip ratio were associated with a higher risk of the primary outcome in women (Figure 1). However, after additional adjustment for LBM, none of these variables remained independently associated with the primary outcome, except for waist-to-hip ratio (Figure 1). In contrast, the associations between LBM and the primary outcome remained robust overall in both sexes, even after adjustment for other anthropometric variables, with the exception of weight in men and waist circumference in women (Figure 1). Details of multivariable-adjusted associations between anthropometric measures and the primary outcome in men and women are presented in Supplemental Tables 5 and 6.

Figure 2 shows the dose-response relationship of the primary outcome with fat mass and LBM in men and women

Table 2 Multivariable associations between lean body mass and the incidence of the primary and secondary outcomes (second-degree AV block, third-degree AV block, and AV block–related pacemaker implantation) in men and women

Lean body mass (per 1-SD increase)*	No. of events	HR (95% CI)†	*P* value	*E* value of estimates	*E* value of LL
Men (n = 176,350)					
Primary outcome	1519	1.15 (1.09–1.21)	<.001	1.57	1.40
Secondary outcomes					
Second-degree AV block	716	1.16 (1.08–1.26)	<.001	1.59	1.37
Third-degree AV block	916	1.14 (1.06–1.22)	<.001	1.54	1.31
AV block–related pacemaker implantation	877	1.17 (1.09–1.25)	<.001	1.62	1.40
Median follow-up period of 11.8 y (quartile 1: 11.1 y, quartile 3: 12.5 y)					
Women (n = 194,065)					
Primary outcome	677	1.13 (1.05–1.22)	.001	1.51	1.28
Secondary outcomes					
Second-degree AV block	304	1.13 (1.01–1.27)	.031	1.51	1.11
Third-degree AV block	408	1.15 (1.04–1.26)	.005	1.57	1.24
AV block–related pacemaker implantation	396	1.19 (1.08–1.31)	<.001	1.67	1.37
Median follow-up period of 11.9 y (quartile 1: 11.2 y, quartile 3: 12.6 y)					

The *primary outcome* is defined as a composite of second- or third-degree AV block.

AV = atrioventricular; CI = confidence interval; HR = hazard ratio; LL = lower limit of the confidence interval; SD = standard deviation.

*1 SD of lean body mass is equivalent to 7.7 kg in men and 5.0 kg in women.

†The main model was adjusted for age, white ethnicity, hypertension, diabetes mellitus, dyslipidemia, coronary heart disease, heart failure, atrial fibrillation, chronic kidney disease, sleep apnea, hypothyroidism, hyperthyroidism, smoking history, alcohol history, high-sensitivity C-reactive protein, physical activity, Townsend deprivation index, education attainment, and history of antiarrhythmic drug use.

after mutual adjustment. In men, fat mass was not associated with the risk of the primary outcome. In contrast, higher LBM was associated with an increased risk of the primary outcome (Figure 2A). In women, an increase in fat mass was not associated with a higher risk of the primary outcome; instead, a decrease in fat mass was associated with a higher risk. In contrast, higher LBM was associated with an increased risk of the primary outcome (Figure 2B).

LBM and electrocardiographic parameters

LBM per quartile increase was associated with a higher prevalence of a prolonged PQ interval (>200 ms) in both men

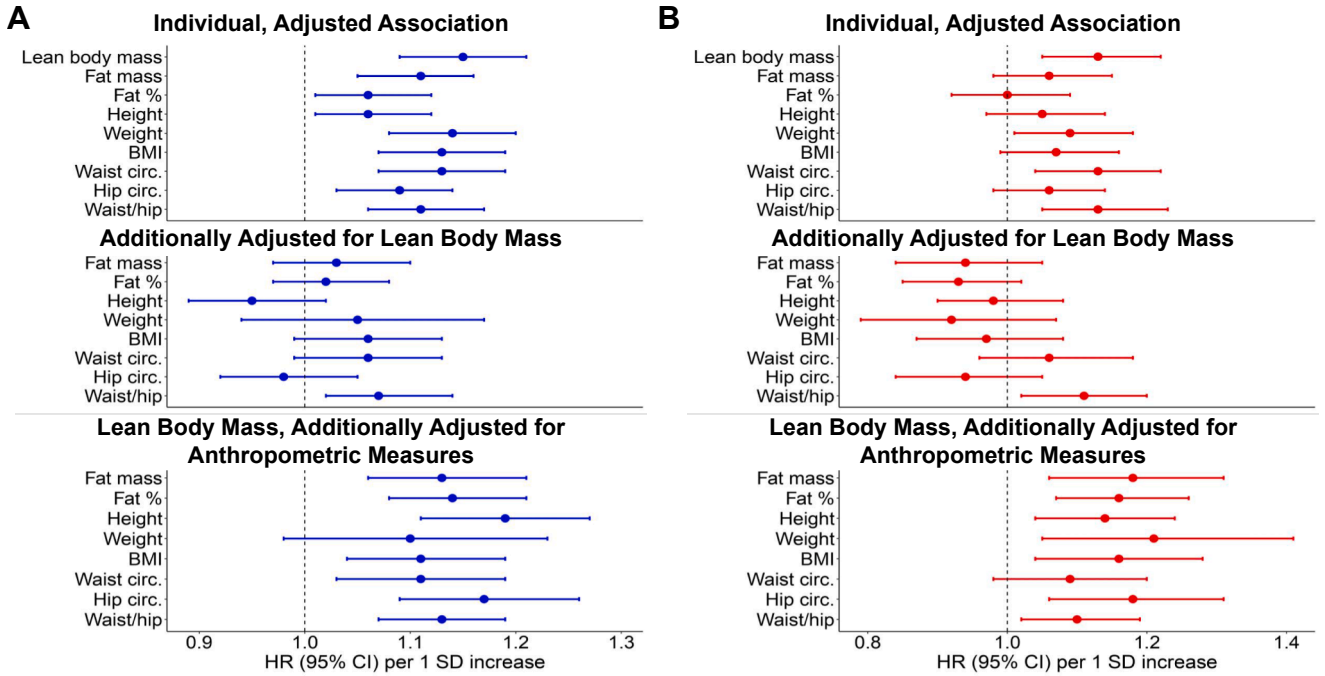

Figure 1 Forest plots of the multiply-adjusted risk of the primary outcome according to lean body mass in (**A**) men and (**B**) women. The *primary outcome* is defined as a composite of second- or third-degree atrioventricular block. HRs per 1-SD increase and corresponding 95% CIs associated with the various anthropometric exposures are shown above. BMI = body mass index; CI = confidence interval; circ. = circumference; HR = hazard ratio; SD = standard deviation.

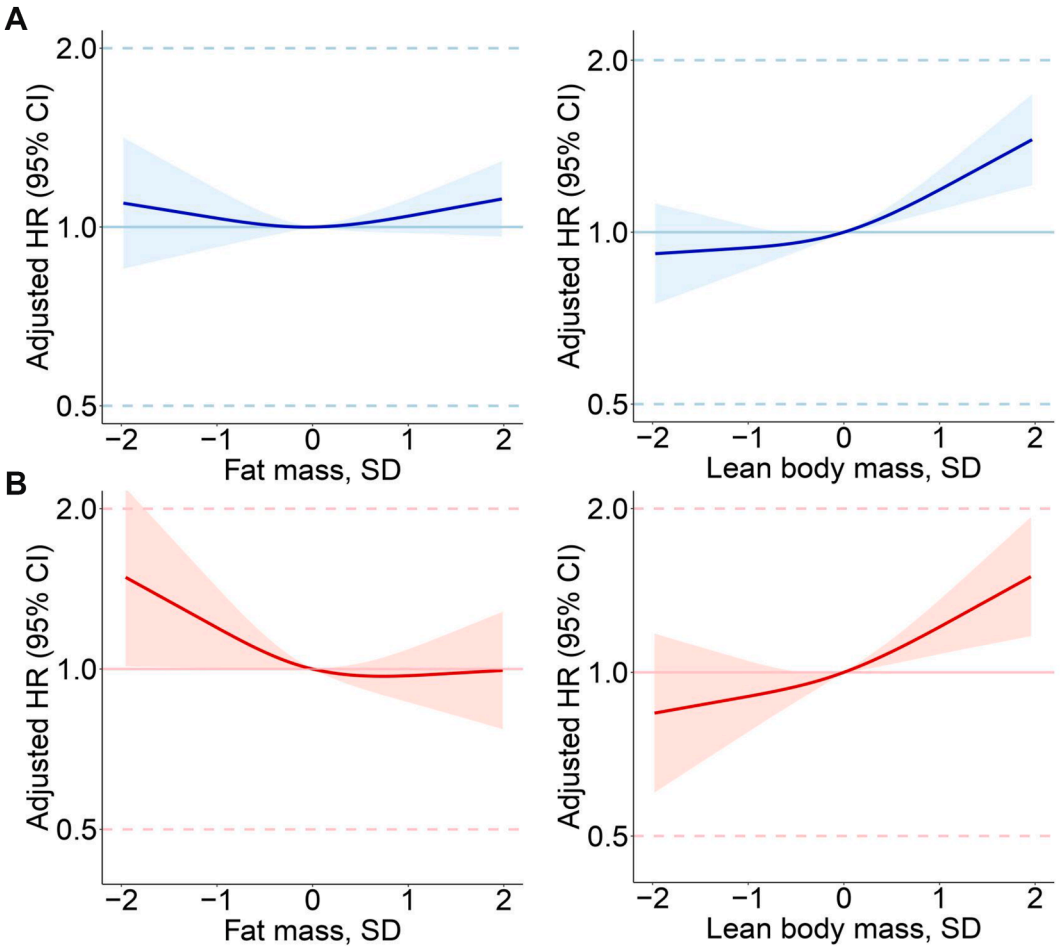

Figure 2 Dose-response relationship of the primary outcome with fat mass and lean body mass in (**A**) men and (**B**) women, after mutual adjustment. The *primary outcome* is defined as a composite of second- or third-degree atrioventricular block. The *solid line* indicates a restricted cubic spline curve, and the *shaded area* indicates the 95% CI. Restricted cubic spline models were fitted using Cox proportional hazards models adjusted for covariates included in the main analysis and mutually adjusted for fat mass or lean body mass. CI = confidence interval; HR = hazard ratio; SD= standard deviation.

and women (P for trend $<$ .001 for all comparisons) (Figure 3). The association between LBM per quartile increase and the prevalence of a prolonged QRS duration (>120 ms) in both sexes is illustrated in Supplemental Figure 3.

LBM and sex differences in the primary outcome

The incidence of the primary outcome was higher in men than in women (Figure 4). The difference in the incidence of the primary outcome between men and women was attenuated after adjusting for sex-related differences in LBM (Figure 4).

Sensitivity analyses

The measurement of LBM derived from bioimpedance analysis was strongly correlated with the measurement of LBM derived from DXA in a subset of UK Biobank participants (Pearson correlation coefficient 0.93; 95% CI 0.93–0.94; P $<$ 0.001 in men and Pearson correlation coefficient 0.93; 95% CI 0.92–0.93; P $<$ .001 in women) (Supplemental Figure 4). Study results remained robust when missing data were handled using multiple imputation by chained equations (Supplemental Table 7). Similar results were obtained when (1) excluding participants with atrial fibrillation or antiarrhythmic drug use at baseline (Supplemental Table 8), (2) adjusting for systolic and diastolic blood pressures instead of hypertension (Supplemental Table 9), (3) excluding participants with the incident primary outcome during the first 2 years of follow-up (Supplemental Table 10), and (4) additionally adjusting for myocardial infarction and heart failure as time-updated covariates (Supplemental Table 11). The main results persisted after additional adjustment for resting heart rate (Supplemental Table 12).

Discussion

Principal findings

In this UK Biobank prospective cohort study, an increased LBM was independently and predominantly associated with a higher risk of the incident primary outcome in both men and women. The sex difference in the risk of the primary outcome was attenuated after adjusting for sex-

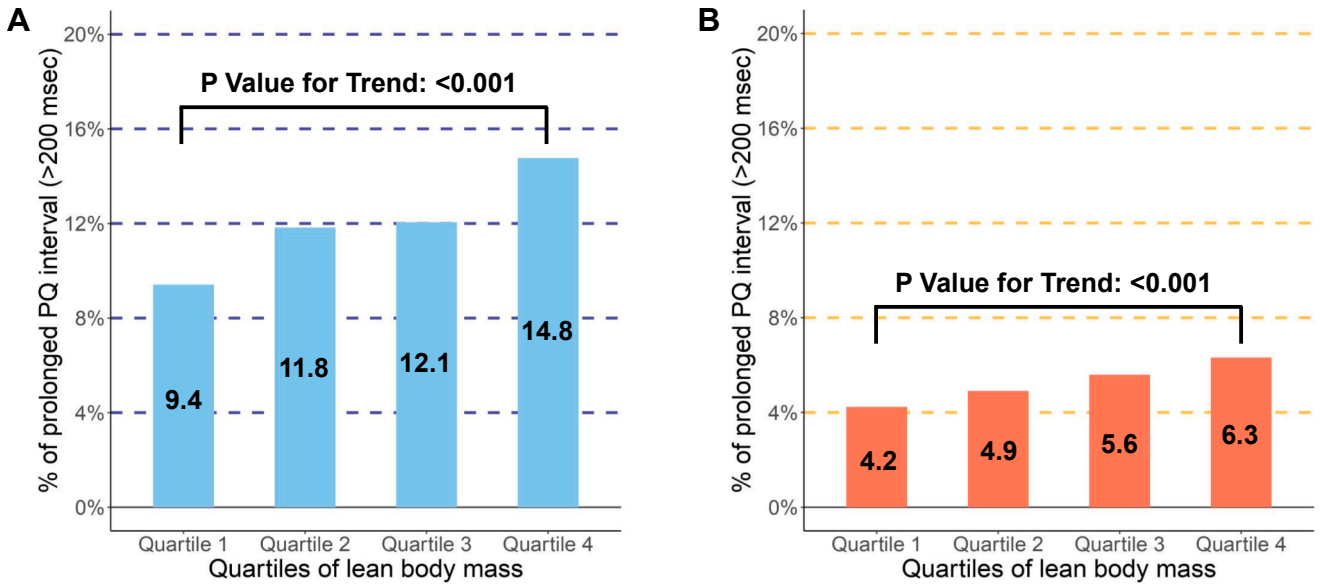

Figure 3 Percentage of the prolonged PQ interval (>200 ms) across quartiles of lean body mass in (**A**) men and (**B**) women. A total of 18,591 participants (9286 in men and 9305 in women) were involved in the electrocardiogram substudy. Participants were stratified into quartiles of lean body mass: for men, quartile 1 (<58.6 kg), quartile 2 (58.7–63.0 kg), quartile 3 (63.1–68.0 kg), and quartile 4 (>68.1 kg); for women, quartile 1 (<41.1 kg), quartile 2 (41.2–43.8 kg), quartile 3 (43.9–46.9 kg), and quartile 4 (>47.0 kg).

related differences in LBM. Higher LBM was associated with a higher prevalence of a prolonged PQ interval.

LBM is independently and predominantly associated with the incident primary outcome

Our study demonstrated that an increase in LBM was independently and predominantly associated with a higher risk of the primary outcome in both men and women, even after adjustment for various anthropometric variables. Notably, the association between LBM and the incident primary outcome was attenuated after adjusting for sex differences in LBM, supporting consistency of our main findings. Our findings suggest that it is LBM, rather than fat mass, that is independently associated with a higher risk of the primary outcome. Notably, elevated baseline high-sensitivity C-reactive protein levels were associated with an increased risk of the primary outcome in both sexes, consistent with previous studies.[4,11] These findings suggest that inflammatory status (eg, metabolic status), rather than fat mass itself, should be considered in both the stratification and the potential modification of AV block risk.[4] The incidence of the primary outcome in the UK Biobank cohort was much lower than that among participants in the Atherosclerosis Risk in Communities Study and Cardiovascular Health Study.[2] The UK Biobank cohort is susceptible to healthy volunteer bias, suggesting that the primary outcome events may be less pathological.[12]

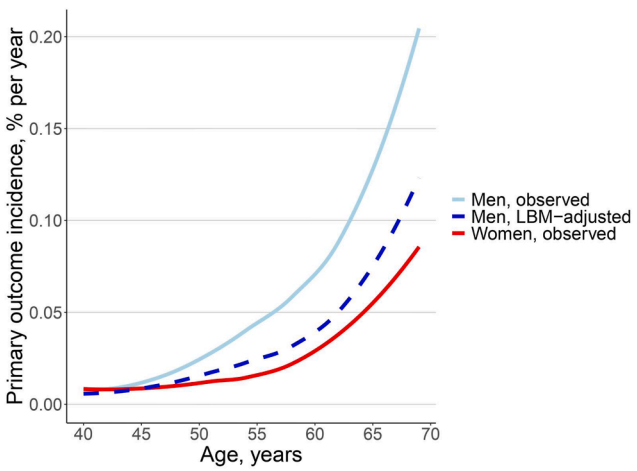

Figure 4 Sex-specific incidence of the primary outcome. The *primary outcome* is defined as a composite of second- or third-degree atrioventricular block. The *solid light blue line* indicates the crude incidence of the primary outcome in men, whereas the *solid red line* indicates the crude incidence of the primary outcome in women. The *dashed blue line* indicates the crude incidence of the primary outcome in men divided by the estimated hazard ratio corresponding to the sex difference in LBM. LBM= lean body mass.

LBM is associated with a prolonged PQ interval

As LBM increased, the prevalence of a prolonged PQ interval also increased, which is a marker of AV conduction delay. However, individuals with higher LBM are often characterized by enhanced parasympathetic tone (vagotonic state).[13] Moreover, individuals with larger body size typically have larger atrial dimensions, which could result in increased conduction distance and thereby partially increase the PQ interval.[14] Whether the prolongation of the PQ interval observed with increasing LBM reflects not only benign vagotonia or physiological structural enlargement but also additional mechanisms remains to be clarified, as the association persisted after additional adjustment for resting heart

rate and was associated with AV block–related pacemaker implantation.

Limitations

Our study has several limitations. First, bioimpedance does not directly measure but predicts LBM, and these predictions can be affected by hydration and body composition heterogeneity. However, the strong correlation between bioimpedance-derived LBM and DXA-derived LBM indicates a low likelihood of misclassification bias. Second, changes in LBM during follow-up after the baseline assessment were not considered when evaluating its association with the incidence of the primary outcome. Third, outcome events were identified using *ICD-10* and *OPCS-4* codes, which may have missed asymptomatic or fatal events because these codes are based on hospital inpatient and outpatient records. Fourth, because of the observational nature of the study, the presence of residual confounding or reverse causality cannot be entirely excluded. Lastly, the majority of UK Biobank participants were middle-aged and of white ethnicity, which limits the generalizability of the findings to other age groups and racial or ethnic populations.

Conclusion

An increase in LBM was independently and predominantly associated with a higher risk of incident AV block in both men and women. Higher LBM was associated with a higher prevalence of a prolonged PQ interval. The observed sex difference in the incidence of AV block was attenuated after adjusting for sex-related differences in LBM. Our findings suggest increased awareness of AV conduction delays, AV block, and the related risk of pacemaker implantation in individuals with high LBM.

Data sharing statement

The data that support the findings of this study are available from the UK Biobank, but restrictions apply to the availability of these data, which were used under license for the present study and are not publicly available. However, data can be obtained from the corresponding author upon reasonable request and with the permission of the UK Biobank.

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Ethics Statement: The present study was conducted under UK Biobank application no. 77793 and approved by the institutional review board of the Yonsei University Health System (approval no. 4–2025–0652). The research reported in this article adhered to the Declaration of Helsinki.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hroo.2026.02.028.

References

1. Kerola T, Eranti A, Aro AL, et al. Risk factors associated with atrioventricular block. JAMA Netw Open 2019;2:e194176.
2. Zhang J, Liu J, Ye M, Zhang M, Yao F, Cheng Y. Incidence and risk factors associated with atrioventricular block in the general population: the Atherosclerosis Risk in Communities Study and Cardiovascular Health Study. BMC Cardiovasc Disord 2024;24:509.
3. Glikson M, Nielsen JC, Kronborg MB, et al. 2021 ESC Guidelines on cardiac pacing and cardiac resynchronization therapy. Europace 2022;24:71–164.
4. Chung HG, Yang PS, Jang E, et al. Metabolic status and atrioventricular block risk: the role of physical activity. Rev Cardiovasc Med 2025;26:37291.
5. Frimodt-Møller EK, Soliman EZ, Kizer JR, et al. Lifestyle habits associated with cardiac conduction disease. Eur Heart J 2023;44:1058–1066.
6. Liu P, Wang Y, Zhang X, et al. Obesity and cardiac conduction block disease in China. JAMA Netw Open 2023;6:e2342831.
7. Chi X, Zhang N, Zhang L, et al. Effects of body mass index and blood pressure on atrioventricular block: two-sample Mendelian randomization. Heart Rhythm 2024;21:174–183.
8. Pan W, Wu Y, Jin C, et al. Association between modifiable risk factors and bradyarrhythmia: a Mendelian randomization study. Heart Rhythm 2026;23:e172–e180.
9. Butler J, Fioretti F, Davies MJ. Redefining obesity: beyond body mass index. Eur Heart J 2025;46:3758–3761.
10. Littlejohns TJ, Sudlow C, Allen NE, Collins R. UK Biobank: opportunities for cardiovascular research. Eur Heart J 2019;40:1158–1166.
11. Wu L, Wu M, Zhao D, et al. Elevated high-sensitivity C-reactive protein levels increase the risk of new-onset cardiac conduction disorders. Cardiovasc Diabetol 2023;22:268.
12. Fry A, Littlejohns TJ, Sudlow C, et al. Comparison of sociodemographic and health-related characteristics of UK Biobank participants with those of the general population. Am J Epidemiol 2017;186:1026–1034.
13. Huston TP, Puffer JC, Rodney WM. The athletic heart syndrome. N Engl J Med 1985;313:24–32.
14. Abhayaratna WP, Seward JB, Appleton CP, et al. Left atrial size: physiologic determinants and clinical applications. J Am Coll Cardiol 2006;47:2357–2363.